JACC Vol. 15, No. 2
February 1990:168A

IMPACT OF FEMALE GENDER ON INCIDENCE AND PROGNOSIS OF INFARCTION-ASSOCIATED HEART FAILURE: THE WORCESTER HEART ATTACK TRIAL.

C Kimmelstiel, M.D., R Goldberg, Ph.D., R Decker, M.D., Z Chen, M.D., M.P.H., V Osganian, P Drady, R.N., J Gore, M.D., F.A.C.C., J Alpert, M.D., F.A.C.C., U. Mass. Med. School, Worcester, MA.

As a part of an ongoing community-wide coronary disease surveillance project involving all 16 hospitals in the Worcester, MA metropolitan area, we examined gender differences in incidence rates (IR) and in-hospital and long-term survival in patients (P) with newly diagnosed infarction-associated heart failure (MICHF). A total of 4109 P with validated MI comprised the overall study population. Results: For the time periods under study (1975, 1978, 1981, 1984 and 1986), females, as compared with males, exhibited a greater IR of MICHF (46% vs. 33%; p<.025). Utilizing a multiple regression analysis, female gender was found to be an independent predictor of MICHF (p<.001). Examining differences in short and long-term survival, P with MICHF had a worse prognosis than P without MICHF (p<.001). However, in P with MICHF, female gender was not found to independently influence either short or long-term survival (p=NS). Conclusion: Following acute MI, women are more likely to develop heart failure than men. Although P with MICHF have a poor prognosis, female gender does not appear to impact directly upon short or long-term survival.

SEX BIAS IN THE MANAGEMENT OF CORONARY ARTERY DISEASE.

Richard Steingart MD, Milton Packer MD, Marrick Kukin MD, Peggy Hamm, Lem Moye' MD PhD, MaryEllen Coglianese RN, Josephine Sollano RN, Sandra Lewis MD, Bernard Gersh MD, Edward Geltman MD, Niki Kantrowitz MD, Stanley Katz MD and Marc Pfeffer MD PhD for the SAVE Investigators. Winthrop-University Hospital, Mineola NY.

In a previous study, we found that physicians referred men (M) 3 times as often as women (W) for catheterization and CABG, even when M and W were matched for the presence and severity of coronary artery disease (CAD). To investigate whether these practices are widespread, we compared the clinical course and treatment that 1578 M, mean age 59 yrs, and 321 W, mean age 62 yrs, received prior to the MI that lead to entry into the multicenter trial of Survival and Ventricular Enlargement. Prior to the index MI, 26.2% of M and 25% of W had angina (pNS). M were more likely to have had a prior MI, 38% M vs 27% W (z=3.4), but the prevalence of chronic CHF was similar (5.7% M vs 6.9% W). W were more likely to be limited by cardiac symptoms than M (only 50% of W were NYHA class I vs 69% of M, z=6.43), but the use of antianginal and anti-CHF drug therapy was identical among M and W. Further, despite greater disability, W were less likely to have cardiac catheterization (27.2% M vs 16.8% W, z=3.91) and CABG (12.9% M vs 7.2% W, z=2.87). Multivariate analyses controlling for age, prior MI, angina, NYHA class, risk factors and drug therapy demonstrated a M to W odds ratio of 1.64 for catheterization and 1.67 for CABG. Conclusion:These data support the existence of sex bias in the management of patients with CAD. Greater attention must be given to the diagnosis and treatment of CAD in W since current strategies result in more residual disability from CAD among W than M.

TYPE A BEHAVIOR AND MALIGNANT VENTRICULAR ARRHYTHMIA.

Barry Miller, Ph.D., Mina Kim, Roger A. Marinchak, M.D., F.A.C.C., Peter R. Kowey, M.D., F.A.C.C., Medical College of Pennsylvania, Philadelphia, PA.

In order to investigate the relationship between Type A behavior patterns and malignant ventricular arrhythmias, we administered 3 validated paper and pencil personality measurements to 3 groups of 10 men (mean age 61 years) who had had a myocardial infarction: Group 1 had suffered a spontaneous episode of sustained ventricular tachycardia (VT) or fibrillation (VF) remote from their infarction, Group II had nonsustained VT on monitoring, and Group III had no repetitive forms. Groups were matched for age and severity of left ventricular dysfunction. Tests used were the Bortner Type A Rating Scale, the Framingham Type A Questionnaire, and the Type A Self-Report Inventory. A repeated measures multiple analysis of variance was performed to determine the differences among the groups for all 3 tests. Each test registered a difference among the groups, with the Group 1 patients having the highest and Group III patients the lowest score ($p<0.046$). Post hoc Tukey tests revealed an inter-group difference only for the Type A Self-Report Inventory ($p<0.002$), a test that is more sensitive to hostility and anger. Thus, patients who develop VT or VF after myocardial infarction may be more prone to these arrhythmias because of a Type A personality profile. Feelings of hostility and anger may render these patients particularly susceptible to a lethal arrhythmia.

Wednesday, March 21, 1990
10:30AM–12:00NOON, Room 06
Unstable Angina

EVIDENCE FOR ACTIVATED CIRCULATING MACROPHAGES/MONOCYTES IN UNSTABLE ANGINA

S.Tanveer Rab, M.D., R. Wayne Alexander, M.D., F.A.C.C., Aftab A. Ansari, M.D., Ph.D. Departments of Medicine & Pathology, Emory University School of Medicine, Atlanta, GA.

There is increasing evidence for immunologic/inflammatory processes (imm/inf) involved in the pathogenesis of coronary artery disease (CAD). We postulated that acute ischemic syndromes represent activation of imm/inf and that this would be reflected in systemic markers of imm./inf. Peripheral blood was collected from 14 patients with unstable angina (UA) and angiographically documented CAD, 25 normal controls, and 25 patients with idiopathic cardiomyopathy (ICM). Using monoclonal antibodies and Microflurometric techniques, T & B lymphocytes and monocytes/macrophages (mono/mac) were determined. There was an increase in cells with the mono/mac Leu-M_3^+ marker and in the mean density (MD) of the MHC class II antigen HLA-DR in patients with UA as compared with those with ICM and normals as indicated:

	N(25)	ICM(25)	UA(14)	P
Leu-M_3^+	10±3	9±4	19±5	<.01
MD HLA-DR	191±27	218±36	387±29	<.01

Thus patients with unstable CAD have increased activated mono/mac. We conclude that unstable ischemic syndromes likely reflect activation of inflammatory processes and that these processes can be followed using easily measured systemic markers.